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Editorial

CURRENT AFFAIRS

STUDENT PARTICIPATION in the affairs of the Society is at a very low ebb and this must be a matter of some concern to the senior members. Is stimulation lacking, are our students too busy with organised courses of training or are they just not interested in the Society?

A few years ago the Natal Branch had a flourishing student group which organised its own affairs. Its photographic group made an 8 mm. colour film on aspects of laboratory life which was very well received when shown to the members of the Branch. Debates and quiz evenings were organised and a host of social activities were held.

In our contemporary journals we find short articles by student members of their respective organisations. One student investigated the effects of delay in handling of specimens on the results in biochemical investigations, another reported on staining to show sex chromosomes. Short articles both, but showing interest and initiative which we are not experiencing here.

We should not allow our students to develop "routine" minds and a great deal can be done by our senior members to encourage students to participate in the wider life of Society.

COMPATIBILITY TESTING IN BLOOD TRANSFUSION

by G. A. I. NEWELL

Natal Blood Transfusion Service, Durban

INTRODUCTION

THE COMPATIBILITY TEST or cross-matching test, as it is sometimes called, is one of the most important tests performed in a laboratory of any kind. From the time blood is withdrawn from a donor until the time it is transfused to a patient numerous tests are performed, the main object of which is to ensure that the correct blood is transfused to the patient. A single mistake anywhere during this process, whether technical or clerical, could lead to a haemolytic transfusion reaction and the death of a patient. Even if the patient did not die, the transfused red cells would be rapidly destroyed, with a poor therapeutic result.

THE PURPOSE OF THE COMPATIBILITY TEST

The purpose of the compatibility test is to discover whether the donor's red cells will react with any specific iso-antibody in the recipient's serum.

The most important of these are the naturally-occurring anti-A and anti-B in the ABO system. Other antibodies such as anti-D and anti-c may have arisen as a result of immunization due to previous blood transfusions, intramuscular injections of blood, or pregnancy. Antibodies such as anti-Le^a, anti-Le^b, anti-P and anti-M (Stratton and Renton, 1958) have occurred in persons who do not appear to have been previously immunised.

Although a sample of donor's cells may be compatible with the serum of the recipient, the recipient may become immunised to those cells. For example, if Rh positive blood is transfused by mistake to an Rh negative person, the cross-matching test will only detect errors in the Rh typing of donor and recipient if the recipient's serum contained Rh antibodies.

PROCEDURE AND TECHNIQUE OF THE CROSS-MATCHING TEST

The technique of the cross-matching test should be distinguished from the procedure by which it is carried out. The technique consists of the actual serological method which is used, e.g., the anti-globulin test, the saline agglutination test, etc., while the procedure embraces all those steps which are taken to ensure that the technique is carried out in the right manner, using the correct samples of blood from donor and patient,

that the results are properly recorded and that the bottle of blood which is despatched to the patient is in fact intended for that patient. For example, in this Service as part of the procedure, the ABO groups of both donor and recipient are checked by a second person to eliminate possible errors in the original grouping.

A good procedure is vital in the cross-matching test, since incompatible transfusions occur more frequently from failure in the procedure than from failures in the technique.

Since the discovery of the ABO Blood group system by Karl Landsteiner in 1900, many tests have been devised to reveal the incompatibility of donors and recipients. At first a very simple procedure was adopted. This merely consisted of placing a drop of donor's cells and patient's serum on a slide; if no agglutination took place the blood was said to be compatible. In the last 15 years, however, with the discovery of several new blood group systems, the development of new techniques has shown that the time-honoured methods for the direct matching of blood prior to transfusion are inadequate and may be extremely dangerous.

It may be said briefly that the cross-matching test consists of testing, by various methods, the patient's serum against the cells of each donor. The following are the most important of these techniques:

1. Tile Method:

This was probably the first method used for compatibility testing. Patient's serum and donor's cells are mixed on a glass or porcelain tile, left to stand at room temperature and read after fifteen minutes.

2. Diamond Open Slide Method:

Patient's serum and donor's cells are mixed on a glass slide on top of a Diamond box. The slide is gently rocked and readings taken after five minutes.

3. Godwin and McCall's Slide Method:

This is a modification of the original Diamond method in that the slide is incubated at 37°C for 15 minutes, and the donor's cells are suspended in 20% albumin (Godwin and McCall, 1949).

4. Saline Tube Method:

Patient's serum and a suspension of donor's cells are placed in a test tube. The tubes may be left at room temperature or at 37°C for 1½ hours.

5. Tube Method with Albumin (Method 1):

As in the saline tube method, serum and cell mixtures are placed at 37°C for 1½ hours. The tubes are then centrifuged and the supernatant saline removed and replaced by an equal volume of 20% bovine albumin. The tubes are replaced in the incubator for a further 30 minutes.

6. Tube Method with Albumin (Method 2):

Serum and cell mixtures are placed in a test tube and one drop of 30% albumin is added. The tube is incubated at 37°C for 1½ hours and centrifuged. In our own laboratory we titrated several batches of anti-D with this method and found no significant decrease in titre if the tubes were incubated for five minutes only (unpublished observation). This method was subsequently used as an emergency procedure.

7. Indirect anti-Globulin Test:

Equal quantities (usually two drops) of patient's serum and a 2–5% suspension of donor's cells are incubated at 37°C for 1 hour. After incubation, the cells are washed four times with isotonic saline, and two drops of anti-globulin reagent are added. After standing at room temperature for five minutes, the tubes are centrifuged at 1,000 r.p.m. for two minutes.

8. The Bromelin Test:

Since the discovery of Bromelin, which is an enzyme derived from pineapple stems, it has been widely used for the detection of incomplete antibodies, and has also been used as a standard cross-matching procedure (Pirofsky, 1959).

Two drops of patient's serum, one drop of Bromelin (0.5% solution) and one drop of a 4% suspension of donor cells, are placed in a test tube at room temperature for 15 minutes and then centrifuged. Results may be read after five minutes in emergency.

This procedure has been highly successful in demonstrating antibodies directed against the ABO, MNS, P, Rh-Hr, Kell, Kidd, Duffy, Lewis, Vel and Sutter blood groups (Gray and Madison, 1959; Pirofsky and Melvin, 1959).

Incomplete Rh and Kell antisera have been shown to produce a spontaneous agglutination of red cells suspended in saline which have been treated with Bromelin (Gray and Madison, 1959).

THE SELECTION OF TECHNIQUE IN THE CROSS-MATCHING TEST

Various factors should be considered when selecting the techniques to be used for the cross-matching test. The experience of the person performing the test and the sensitivity of the technique in the detection of anti-A, anti-B and other blood group antibodies constitutes two important criteria. The test should be conducted with reasonable speed, should be relatively simple to perform and be reliable. What then are the best methods for routine use?

The tile method was shown by Dodge to give as much as 61.8% false negative results, and with the Diamond slide method 37.4% false negative results were obtained (Dodge, 1952). The two modifications

of the Diamond slide method were found to be suitable for rapid work and reasonably reliable, giving less than 4.0% false negative results (Dodge, 1952). These methods would be useful to technologists in outlying hospitals where a cross-matching test is performed only as an incidental procedure.

It has been found advantageous to use the saline tube method at room temperature rather than at 37°C, as many as 2–3% of anti-A and anti-B antibodies may fail to show decisive agglutination at 37°C, and cold specific atypical antibodies such as anti-P and anti-M will also be readily detected at this lower temperature.

The two tube methods with albumin have been found to compare satisfactorily with the anti-globulin test (Dodge, 1952), but should not replace the saline room temperature method as errors in the ABO grouping may go undetected (Jennings, 1954).

In the hands of skillful technicians, the anti-globulin test is considered to be the test of choice. It is widely effective in detecting all kinds of incomplete blood group antibodies if the technique is correctly carried out. The commonest errors are failure to wash the cells adequately, failure to wash the slide or tube with which the anti-globulin serum comes in contact, and the use of the anti-globulin serum in an inappropriate dilution. The greatest disadvantage of this test is that it is time-consuming.

The advantages of the Bromelin technique are its simplicity and the short incubation period. It has, however, one great disadvantage in that it has been found to give negative reactions against some weakly reacting Kell and Duffy antibodies.

A tube test is recommended rather than a tile technique. Since no single test will detect both complete and incomplete antibodies with complete reliability, it is desirable to incorporate at least two different techniques in the standard cross-matching procedure. It is unlikely that the Bromelin test will replace the anti-globulin test, but, used in parallel, they will undoubtedly be highly effective in detecting all blood group antibodies known to date.

Thanks are due to Dr. B. G. Grobbelaar, Medical Director of Natal Blood Transfusion Service, for permission to publish this article.

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RHESUS INHERITANCE

by

F. A. WARD

Natal Blood Transfusion Service

THERE ARE two main methods of Rhesus notation in use throughout the world to-day. One is the CDE method evolved by Fisher, Race, and Cappell, and is often referred to as the British system. The other is the Rh-Hr method of Wiener, which is also known as the American system.

It is often said that it does not matter a straw which system is used provided the user and his listener understands what is meant. This tolerant attitude, as we will soon see, is to be deplored. The choice between the Wiener and Fisher systems is not analogous with the choice between the Goren and Culbertson system at the bridge-table, it is more akin to the choice between the medieval view that the earth was flat and the modern view that it is round.

Fisher's notation was conceived, and developed on the basis of his theory of triple inheritance; Wiener's notation, on the other hand, is based on Wiener's theory of multiple alleles. To use Fisher's notation therefore implies a belief in the theory of triple inheritance while to use Wiener's implies a belief in the theory of multiple alleles.

The choice, therefore, is not merely a choice between two systems of nomenclature, but between two modes of Rhesus inheritance. If Wiener's theory is correct, then his system must be used; similarly if Fisher is correct, then Fisher's nomenclature is to be preferred. These two views of Rhesus inheritance are mutually exclusive, and attempts to harmonize them lead to mental confusion if not insanity.

In all scientific enquiry it is necessary to begin with undisputed fact. Both Fisher and Wiener would agree that there are five antisera available which probe the problem. These antisera are named as follows:—

Wiener:	Anti-**Rh_o**	anti-**rh′**	anti-**rh″**	anti-**hr′**	anti-**hr″**
Fisher:	Anti-D	anti-C	anti-E	anti-c	anti-e

With these antisera we find that the most common reaction pattern among Caucasians is:—

Anti-**Rh_o**	anti-**rh′**	anti-**rh″**	anti-**hr′**	anti-**hr″**
Anti-D	anti-C	anti-E	anti-c	anti-e
+	+	—	+	+

Fisher would call this blood CDe/cde, while, Wiener would call it Rh_1rh.

This calls for the first critical comment. The presence of the little "d" in the second part of Fisher's symbol implies the presence of the d antigen. This is unscientific as there is no anti-d serum in existence and hence the d antigen has never been demonstrated.

Another common Rhesus type gives the following reaction:—

Anti-**Rh_o**	anti-**rh′**	anti-**rh″**	anti-**hr′**	anti-**hr″**
Anti-D	anti-C	anti-E	anti-c	anti-e
—	—	—	+	+

Fisher would call this blood cde/cde, and Wiener would call it rh. A further criticism can be added here. The Wiener symbol is much shorter than the Fisher symbol.

If the two Rhesus types given above are mated, we would often find a set-up which Fisher would explain:—

Father CDe/cde X Mother cde/cde

Child CDe/cde

and Wiener would write:—

Father Rh_1rh X Mother rh

Child Rh_1rh

It will be noted that according to Fisher, three characters are inherited from the father, namely C, D and e and three from the mother, namely c, d and e.

Wiener, on the other hand, would say that the child inherited the gene R^1 (which determines the character Rh_1) from the father, and the gene *r* (which determines the character rh) from the mother.

Thousands of family studies similar to the above show that the characters symbolised by the letters CDe (Rh_1) are always inherited as a unit. Wiener would find no reason to comment on this as he says that only one gene was inherited from the father. Fisher, on the other hand, explains this block inheritance by saying that the three genes, C, D and e are closely linked.

At this point an important criticism is made against Fisher's view. If three separate genes are inherited, then the phenomenon of crossing-over would be expected to occur. But crossing-over has never been observed. Some of Fisher's protagonists say that perhaps crossing-over does occur and that it accounts for some of the less common Rhesus types, but it has never been demonstrated by family studies.

Allowing for the principle of crossing-over and considering the family already mentioned, children of the following Rhesus groups are possible:

CDe/cde, cde/cde, cDe/cde, Cde/cde.

That is to say, according to Fisher's view, four different Rhesus groups are possible. According to Wiener however, only two Rhesus types are possible in this family, namely Rh_1rh, and rh. In other words, those

who hold Fisher's view must admit of this mating being capable of producing two kinds of children which are precluded by Wiener's view. Put in other way, this means that in a case of disputed paternity, it might be possible to exclude paternity if the situation is viewed through Wiener's eyes, but this would not be possible according to Fisher's view.

We have now reached a stage where we see that the choice of theory of Rhesus inheritance is not merely of academic, but of real practical medico-legal importance. Those therefore, who have already decided on a system of nomenclature may have second thoughts about it now that some of the implications involved are appreciated. It is again emphasized that the choice between these two systems is not merely a choice between two nomenclatures but a choice between the theory of multiple alleles and the theory of triple inheritance by linked genes.

It is now necessary to look into these theories more closely and thus try to decide which one is more in harmony with the facts of Nature.

THE THEORY OF MULTIPLE ALLELES

In order to grasp this theory it is necessary to be quite clear about the meaning of four terms, namely phenotype, genotype, agglutinogen and factor.

1. *Rhesus phenotype*

This is merely a statement of the reactions of a blood specimen recorded according to agreed principles. The phenotype Rh_1rh means that these red cells reacted in the following way:

Anti-**Rh**$_o$	anti-**rh**′	anti-**rh**″	anti-**hr**′	anti-**hr**″
+	+	—	+	+

2. *Rhesus genotype*

This is a statement of the genes an individual inherited from his parents. It is determined by family studies but sometimes it can be determined by direct Rhesus testing. An individual who is phenotypically Rh_1rh may be genotypically R^1r or he may be R^oR^1 or R^or^1. Which he is would have to be decided by family studies. On the other hand, an individual whose red cells give the following reaction:

Anti-**Rh**$_o$	anti-**rh**′	anti-**rh**″	anti-**hr**′	anti-**hr**″
—	—	—	+	+

is phenotypically rh and genotypically *rr*.

If this is difficult to understand, consider a more familiar example. An individual who is phenotypically A may be genotypically *AA* or *AO*. Which of these he is would have to be decided by family studies. But an individual who is phenotypically O must be genotypically *OO*, and an individual who is phenotypically AB must be genotypically *AB*. Similarly there are thirteen circumstances in which the Rhesus phenotype is a direct reflection of the Rhesus genotype.

3. *A Rhesus Agglutinogen*

This is a substance on the surface of red cells. There are many kinds of Rhesus agglutinogens, each being determined by the Rhesus genes inherited. Thus if a person inherits the gene R^1 he will possess the agglutinogen Rh_1. Agglutinogens are characterised by the Rhesus factors which they possess and also by those they lack.

4. *A Rhesus Factor*

Rhesus factors are attributes of Rhesus agglutinogens and determine their serological reactions. We do not know the precise nature of these factors, or how many of them there are on Rhesus agglutinogens, but on the commonly occurring agglutinogens there are three known factors. Thus:

Rhesus agglutinogen Rh_1 contains factors **Rh_o**, **rh′** and **hr″**.
Rhesus agglutinogen Rh_2 contains factors **Rh_o**, **rh″** and **hr′**.
Rhesus agglutinogen rh contains factors **hr′**, **hr″** and **hr**.

No two different Rhesus agglutinogens have the same set of Rhesus factors (if they did, they would not be different) but they may have factors in common. Agglutinogen Rh_1 and agglutinogen Rh_2 each have factor **Rh_o** in common, but nevertheless each has a different set of factors.

If there is still difficulty in appreciating the difference between a Rhesus agglutinogen and a Rhesus factor, the following analogy may help. We all agree that lactose and glucose are sugars. They both reduce Benedict's solution (hence they have at least one factor in common) but glucose is capable of being fermented by yeast whereas lactose is not. (Hence there is at least one factor which is not shared in common.) Similarly we all agree that Rh_1 and Rh_2 are Rhesus agglutinogens. They are both agglutinated by the reagent anti-**Rh_o**, therefore they have one factor in common (which we call **Rh_o**). But agglutinogen Rh_1 is capable of being agglutinated also by a reagent called anti-**rh′** whereas Rh_2 is not (hence there is at least one factor which is not shared by both agglutinogens).

To carry the analogy one step further—just as glucose is identified by its factors (which we usually call properties) so also is Rh_1 identified by its factors (which we might well call properties). Just as glucose cannot be identified by its ability to reduce Benedict's solution alone, neither can Rh_1 be identified by its reaction with anti-**Rh_o** alone. Thus, just as glucose is identified by a *set* of properties so Rh_1 is identified by a set of factors.

According to Wiener's convention, ordinary type should be used for Rh agglutinogens and bold-faced type for Rh factors and anti-sera. Furthermore italics should be used for genes and genotypes. When writing by hand or with a typewriter, genes and genotypes should have a single underline, while factors and antibodies should have a double underline.

Having gained a clear idea of the meaning of the above terms we must now consider some family pedigrees which throw light on the subject. Tom, Dick and Harry were three Rhesus positive men (by which is meant that their red cells agglutinated when mixed with anti-**Rh_o** serum) who were married to three Rhesus negative women (by which is meant that their red cells did not agglutinate when mixed with anti-**Rh_o** serum). Tom was not only Rhesus positive, more specifically he was of type Rh_o.

Tom's family set-up

Mrs. Tom	Tom	Children			
		1	2	3	4
rh	Rh_o	Rh_o	rh	Rh_o	rh

In this family we see that two children had the same Rhesus type as Tom and two had the same as Mrs. Tom. From this family, therefore, we learn of the existence of a gene which determines the group rh (which we will agree to call *r*) and a gene which determines Rh_o (which we will agree to call R^o). Thus we see that there are at least two Rhesus genes.

Dick's family set-up

Mrs. Dick	Dick	Children			
		1	2	3	4
rh	Rh_1	Rh_1	rh	Rh_1	rh

From this family we learn of the existence of another gene—the one determining agglutinogen Rh_1. We will agree to call this gene R^1.

Harry's family set-up

Mrs. Harry	Harry	Children			
		1	2	3	4
rh	Rh_1	rh′	Rh_o	rh′	rh′

This is the kind of family set-up that makes the gossips sit up. At sight it looks as though Mrs. Harry knew Tom, otherwise where did the second child come from? The question of adultery need not arise. Harry though phenotypically Rh_1, was genotypically R^or'. Hence the children rh′ and Rh_o. This family proved the existence of yet another gene, namely, *r*′.

Other family studies prove the existence of further genes.

Mrs.	Mr.	Children			
		1	2	3	4
rh	rh″	rh	rh″	rh″	rh

This family proves the existence of a gene *r*″.

Mrs.	Mr.	1	2	3	4
rh	Rh_2	Rh_2	rh	rh	Rh_2

Therefor, there is a gene R^2.

Mrs.	Mr.	1	2	3	4
rh	Rh_1Rh_2	rh	Rh_1Rh_2	rh	Rh_1Rh_2

This family is unusual. The father transmitted his unusual group to his second and fourth children. Therefore, there is another gene which is called R^z.

Mrs.	Mr.	1	2	3	4
rh	rh′rh″	rh	rh′rh″	rh	rh′rh″

Again the father transmitted his unusual blood to his second and fourth children. Therefore, there is a gene determining this character which by convention we called r^y.

Similar family studies have shown the existence of genes determining other Rhesus types. The following genes are now known:
$r, r', r^w, r'', r^y, R^o, R^1, R^{1w}, R^2, R^z, r^v, r^G, R^{ov}, R^{oa}, R^{ob}, \overline{R^o}, R^{1a}, R^{1b}, R^{2a}, R^{2b}$,
and even that is not all. This list does not include the genes which determine the Rh_o variants.

The point however, has been made, namely that there are many allelic genes any two of which determine the Rhesus reactions of the blood of individuals.

THE THEORY OF LINKED GENES

It might be thought that the concept of an agglutinogen was not necessary on the grounds that these various Rhesus factors are determined by genes directly without the intermediary of an agglutinogen. If this were so, we should have to postulate the existence of genes determining the factors, **hr′** and **hr″**. Having made this assumption, we should then be faced with another question. Why do these factors never occur alone but always in association with other factors? To answer this question, we should have to postulate further that the genes determining these factors are linked to the genes determining the other factors, with which they are associated. Thus **hr″** is often found in association with **Rh_o** and **rh′**, hence we would say that the genes determining these factors are linked. In other words; if D = the gene determining **Rh_o**, and C = the gene determining **rh′**, and e = the gene determining **hr″**, then D, C and e are linked. And that is exactly what Fisher says.

To put it graphically:

Father	Mother
CDe/cde	cde/cde

Child
CDe/cde

Fisher would say that the genes C, D and e in the father, being linked were passed on to the child as a single unit.

If however, this theory were true then we would expect the phenomenon of crossing-over to take place from time to time. In other words, we would expect to find (for example) this:

Father		Mother
CDe/cde		cde/cde
	Child	
	cDe/cde	

But despite thousands of family studies similar to Dick's, this set-up has never been found.

The failure of crossing-over to occur is the most telling argument against the theory of linked genes. For this and other reasons, Wiener having considered it, discards this explanation of Rhesus inheritance in favour of the more likely theory of multiple alleles.

And yet we cannot look at Rhesus inheritance without a feeling that there is a *linkage* somewhere or other. These feelings are satisfied when it is recalled that the Rhesus factors are linked to the Rhesus agglutinogens. There is therefore, no need to assume that Rhesus genes are linked to each other.

To say that the sun rises in the morning and sets ine th evening is now known to be nonsense. Yet this erroneous view of solar movement satisfied the minds of men for centuries and even in to-day's world of space travel, it will suffice for most practical purposes. No doubt even Gagarin and Shepard enjoy a beautiful sunset just as much as we earth-bound mortals. Similarly the concept of linked genes will suffice for most practical puposes and indeed for all practical purposes if we remain at a superficial level, but as we go deeper and deeper into the complexities of the Rhesus system, we find that what at first sight appeared to be a reasonable, simple and ordered view, does not accord with the facts.

This paper could not have been written without the help of Dr. Wiener's books, articles and personal letters. To him therefore, I am indebted for any merit in this work.

INSTRUMENT NEWS

1. *Radio-Iodine Monitor NE* 8421:

This monitor is fully transportable and has a head unit consisting of a transistorised preamplifier and NE 5502 lead castle with 2″ photomultiplier and $1\frac{1}{2}$″ x 1″ sodium iodide crystal. The analyser unit is similar in general design to the Nuclear Enterprises Mark XII Scintillometer with three channels arranged to cover the neighbourhood of the main I^{131} gamma photopeak and to produce a difference count which is a sensitive function of the amount of I^{131} even in the presence of strong competing gamma radiation from other iodine activities. The control unit is constructed with plug-in boards, is fully transistorised and completely shock mounted for vehicle operation. The unit has obvious application to other monitoring problems.

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NOTICES

THE NATAL PATHOLOGICAL LABORATORY, Chancery Buildings, Smith Street, Durban, requires technical staff. The laboratory is not, at the moment recognised, for the purpose of training and qualified applicants would be preferred. Details of salary, etc., may be obtained from: Dr. Lindsay Walker at the above address or Tel. Durban 24063.

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All contributions are to be addressed to:—The Editor, The South African Journal of Medical Laboratory Technology, c/o Central Pathological Laboratory, Private Bag, Jacobs, Natal.

Contributions may be written in English or Afrikaans, and should preferably be typed in double-spacing on foolscap sheets on one side of the paper only.

Figures should be drawn in Indian ink, and all figures and tables should be labelled as such (e.g Figure 1, Table 1, etc.).

Authors should make adequate references to previous works on their subjects. These should be set out as follows:—Author's surname and initials of Christian names; the year of publication (in parentheses); the name of the journal, which should be abbreviated according to the World List of Scientific Periodicals (see below); the volume number (underlined); and the first page reference.

Example:—Moron, I. B. (1960). *J. unsuccess. Med.*, 20, 99. References to books should give the author's name and initials, the year of publication, title of book, name of publisher, and town in which published.

References should be arranged in alphabetical order of the authors' surnames. If more than one work by the same author is listed, these should appear in chronological order.

Technologists are reminded that regulations demand that all original articles of a technical or scientific nature must be approved by the heads of their departments before being submitted for publication.

Title abbreviations according to World List of Scientific periodicals

All nouns commence with capital letters, and adjectives small letters. Articles, conjunctions and prepositions are omitted.

Examples:—

J. Amer. med. Ass.	*S. Afr. J. clin. Sci.*
Lancet	*Stain Tech.*
Amer. J. clin. Path.	*J. Bact.*

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If requested before publication, 24 reprints of original articles will be supplied free to contributors. As a temporary measure, contributors are asked to defray the costs of publishing diagrams and photographs accompanying articles.

KENNISGEWING AAN INSENDERS

Alle bydraes moet aan die Redakteur, Die Suid-Afrikaanse Tydskrif vir Mediese Tegnologie, Sentrale Patologielaboratorium, Privaatsak Jacobs, Natal, geadresseer word.

Bydraes, in Engels of Afrikaans, moet verkieslik op foliopapeir getik word (dubbel spasiëring). Slegs een kant van die papier moet gebruik word.

Illustrasies moet met oosindiese ink geteken word en tabelle aangedui word (bv. Figuur 1, Tabel 1, ens.).

Bydraes moet voldoende verwysings na vorige werk in verband met hulle onderwerpe aangee. Verwysings moet as volg uiteengesit word:—Outeur se van en voorletters; die jaar van uitgawe (in hakies); die naam van die tydskrif (verkort volgens die Wêreldlys van Wetenskaplike Tydskrifte, sien hieronder); die boekdeelnommer (onderstreep) en die eerste bladsyverwysing.

Byvoorbeeld:—Moron, I. B. (1960). *J. unsuccess. Med.*, 20, 99. Boekverwysings moet die skrywer se van en voorletters, jaar van publikasie, titel, naam van uitgewer en stad waar uitgegee, aandui.

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Tegnoloë word daaraan herinner dat die publikasie van oorspronklike artikels van 'n tegniese of wetenskaplike aard, die goedkeuring van hulle departementele hoofde vereis.

Titelverkortings volgens Wêreldlys van Wetenskaplike Tydskrifte

Selfstandige naamwoorde moet met hoofletters geskryf word en bygoeglike naamwoorde met klein letters. Voorsetsels, voegwoorde en lidwoorde moet uitgelaat word.

Byvoorbeeld:—

J. Amer. med. Ass.	*S. Afr. J. clin. Sci.*
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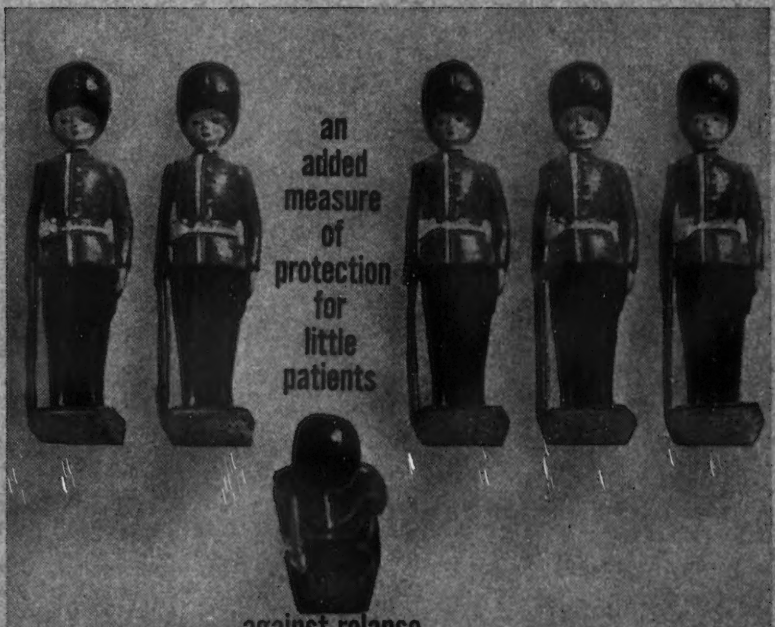

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